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An 8-year-old boy with a 4-day history of fever, cough and malaise, and a 2-day history of painful calves and difficulty walking

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Clinical information

An 8-year-old boy presented in February 2003 with a 4-day history of fever, cough and malaise, and a 2-day history of painful calves and difficulty walking. There was no history of similar episodes. The patient and the family denied recent trauma, unusual physical activity, or a family history of muscle disease. He suffered from an attention deficit/hyperactivity disorder and was on long-term medication with methylphenidate. On examination, the child was alert, afebrile (36.9°C), and normotensive. There was no rash and the heart and lung examinations were normal. The gait was wide-based and high stepping. He had tenderness of the gastrocnemius-soleus muscles with restriction of passive dorsiflexion due to pain (Homan sign). Neurological examination was otherwise normal.

Laboratory investigations gave the following results: haemoglobin 139 g/l, WBC 3.7×10^9 /l, platelets 173×10^9 /l, C-reactive protein 3 mg/l (normal up to 5 mg/l), creatinine 45 µmol/l (normal up to 56 µmol/l), creatine kinase 10,760 IU/l (normal up to 247 IU/l) and aspartate aminotransferase 275 IU/l (normal up to 50 IU/l). The urine was normal in colour and a urine dipstick test was negative. A direct fluorescent antibody staining failed to detect influenza A or B virus and parainfluenza virus on a nasal swab.

Diagnosis: myalgia cruris epidemica (benign acute childhood myositis)

The patient was treated with oral paracetamol for 2 days. Four days later he felt entirely well with no muscle tenderness or weakness.

Discussion

When a young child begins to limp or an older child complains of limb pain, it may be impossible to distinguish muscle pain from joint or bone pain but the clinical and laboratory features noted in this child clearly indicated muscle pains [6].

So-called “growing pains” involve mostly the thigh and calf muscles, mainly at night in bed, but are not associated with difficulty in walking. The peak age of growing pain is 10 to 13 years, and at that age, approximately 20% of boys and 30% of girls complain of them [5, 6]. In Guillain-Barré polyneuropathy, particularly in cases with an abrupt onset, tenderness to muscle palpation is common in the initial stage. In this disease, tendon reflexes are usually lost but are exceptionally preserved until later [4, 6]. Deep venous thrombosis of the calf is often associated with posterior calf tenderness and Homan sign but is mostly unilateral. Furthermore, deep venous thrombosis is rather rare before puberty unless related to characteristic conditions including surgery, trauma or immobilisation [1]. It is worthy of mention, however, that creatine kinase levels in blood are mostly normal in children with “growing pains”, Guillain-Barré polyneuropathy or deep venous thrombosis.

Acute onset of symmetrical calf muscle pain and tenderness, weakness of the lower extremities, inability or refusal to walk in the context of a influenza-like illness, elevated creatine kinase levels but normal urinalysis are the peculiar features of benign acute childhood myositis [2, 3, 7, 9]. This cause of muscle pain and weakness was first reported approximately 40 years ago as myalgia cruris epidemica. It typically affects

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Table 1 Muscle syndromes associated with common viral infections

	Benign myalgia	Benign acute myositis	Myoglobinuric rhabdomyolysis
Age	Any	Mostly children aged 2–10 years	Any
Frequency	Very frequent	Rather rare	Rare
Localisation	Back and proximal extremities	Calves	Diffuse (extremities and trunk)
Course of viral disease	Prodrome or early phase	1–2 days after onset of symptoms, sometimes “recovery phase”	Mostly “recovery phase”
Creatine kinase activity	Normal	Elevated	Extremely elevated
Urinalysis	Normal	Normal	Dark coloured urine, test positive for blood by dipstick, no red cells on on microscopy ^a

^aThe urinary benzidine dipstick does not differentiate between haemoglobin and myoglobin

children aged 2 to 10 years and resolves within 3 to 10 days. Several features differentiate benign acute childhood myositis from juvenile dermatomyositis [3]. Benign acute childhood myositis is a para- or post-infectious process that typically follows influenza or parainfluenza. More rarely, it follows other infectious viral illnesses such as coxsackie and adenovirus, or *Mycoplasma pneumoniae*. Benign acute childhood myositis predominantly affects the distal legs, in particular the gastrocnemius and soleus muscle groups, whereas juvenile myositis and dermatomyositis predominantly affect the proximal muscles. In juvenile dermatomyositis, distinct cutaneous features often precede the muscle disease: violaceous or dusky erythema with oedema in a periorbital distribution and Gottron papules (slightly elevated, violaceous to dusky red papules or plaques over bony prominences, particularly the knuckles) [3].

The spectrum of muscle syndromes linked with viral infections ranges from benign commonly experienced myalgias to rhabdomyolysis with myoglobinuric kidney disease (Table 1) [3, 8]. Mild to moderate diffuse myalgias are frequently reported during the prodrome or early phase of any acute viral infection. The back and proximal extremities are commonly involved and mild muscle tenderness may occur without weakness or laboratory abnormalities suggestive of muscle inflammation. Benign acute childhood myositis is an intermediate muscle syndrome accompanying acute viral infections that is primarily seen in children. At the other end of the spectrum is viral myositis accompanied by massive rhabdomyolysis and its serious consequences. Patients present with a history of a preceding upper respiratory tract infection followed by high fever, diffuse myalgias, and anorexia. Gastrointestinal symptoms may also be present. This prodrome of viral symptoms occurs 1 to 14 days before the onset of rhabdomyolysis. The distribution of muscle involvement is more diffuse than in the intermediate syndrome in which involvement is limited to the legs. Both upper and lower extremities as well as trunk muscles are involved. Some patients present with abdominal pain due to abdominal muscle involvement. Muscles are tender

and may be slack or oedematous. Weakness parallels the severity of muscle involvement. Extreme elevations of creatine kinase can be seen in acute viral myositis with levels up to more than 500,000 IU/l. The urinary findings include a dark coloured urine with distinctive urinalysis features suggestive of myoglobinuria: a positive test for blood with the benzidine dipstick without red cells seen on microscopic examination (the urinary benzidine dipstick does not differentiate between haemoglobin and myoglobin which are structurally related).

Two further causes of acute “myositis” that are unlikely to account for the history of our child include rhabdomyolysis secondary to exertional stress and the use of regular and illegal drugs. Perhaps the most frequent cause of drug-induced myopathy is the administration of statins, very popular lipid-lowering agents [3, 8]. To the best of our knowledge, there are no data relating the use of methylphenidate to a myopathy.

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